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* See especially page 398.

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Hiroshima and Nagasaki: A Commentary

P. J. D. Lloyd

The Japanese findings on the effects of nuclear weapons exploded over Hiroshima and Nagasaki have recently been published and are reviewed here. First, the physical events occurring immediately after the explosions are outlined; then, the short- and long-term effects upon the environment are described, as are the short-, intermediate- and long-term effects upon the populace. The findings are discussed in the light of the common wisdom that the major hazard which arises from nuclear weapons is that of radioactivity. It is pointed out that by far the greatest proportion of deaths and injuries arose from thermal effects; that about 10% of the short- and intermediate-term mortality was caused by the effects of massive doses of radiation; that the long-term effects are detectable above background, but have contributed little additional to the toll of human suffering; and that while the effects of fallout and induced radiation were initially apparent on close examination, no long-term effect has been identified, nor has there been any contribution to mortality or disease from this source. It is concluded that endeavours to resist nuclear weapons may be failing because of irrational attacks upon the long-term radiation effects of these weapons, rather than upon the chemical and biological effects which have already been found totally unacceptable when caused by other types of weapons.

On 6 August 1946, the city of Hiroshima disappeared at precisely 08h15. A uranium bomb of some 13 000 tons of TNT equivalent had exploded at a height of 580 m over the city centre. Three days later, at 11h20, a plutonium bomb of some 22 000 tons of TNT equivalent exploded at a height of 503 m over Nagasaki. Time heals, and distance brings perspective. After thirty-four years the Japanese were able to look with relative dispassion on the months following that August, and have now published their findings.[1] It is the purpose of this article to review the chronicle of their experiences, because it suggests that we may be debating the case for and against nuclear weapons from the wrong position.

Nature of the weapons used at Hiroshima and Nagasaki

The basic physics of the atomic bomb is well-known. Fissile nuclides, typically $^{235}_{92}U$ or $^{239}_{94}Pu$, are compressed into a critical mass; an uncontrolled chain reaction ensues, with the splitting of each nuclide into at least two fragments; neutrons are released to sustain the reaction; some of the mass of the original nuclide is converted into energy. Each gram of uranium which splits releases about 8.2×10^{10} J, so that to create the energy equivalent to about 20 000 tons of TNT (9×10^{13} J) requires the destruction of about 1 kg of $^{235}_{92}U$.

A point source of 9×10^{13} J will reach temperatures of the order of 10^6°C, destroy its confinement, and begin to dissipate energy. A fireball is formed, and the thermal expansion which results gives rise to a shock wave. Initially, the surface of the fireball and shock wave are coincident, but after about 0.1 ms the shock wave separates from the fireball surface; the fireball then has a radius of about 15 m and a temperature of some 300 000°C.

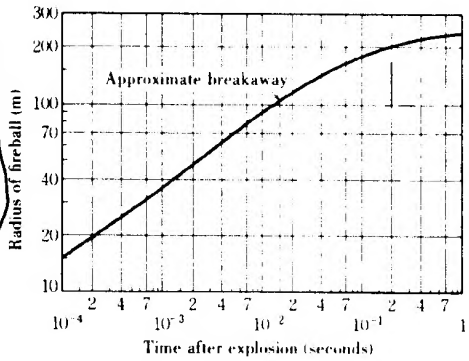

Fig. 1. The growth of the fireball for a 20 kt weapon.

The shock wave spreads and heats the air in its path. The air becomes luminous, and effectively shields the fireball. However, after about 15 ms, the shock wave has travelled so far and dissipated so much energy that it can no longer heat the air to luminous temperatures. The fireball then becomes visible from outside. This is referred to as the 'breakaway' point. The fireball continues to expand and reaches it maximum size of about 220 m one second after the explosion. The growth of the fireball is shown in Fig. 1, and the observed surface temperature of the ball in Fig. 2. The minimum in the observed temperature at the breakaway point is caused by the predominance of high-energy ultraviolet rays during the first 15 ms, and the increase in the near ultraviolet, visible and infrared rays thereafter. A few seconds after the explosion, buoyancy forces come into play and the fireball starts to rise, drawing air in below it, and forming the typical 'mushroom' cloud within a few minutes. The unstable, radioactive elements formed in the fission process tend to be carried up in the fireball, and are precipitated as 'fallout' from a height of 1 km or more as moisture carried into cooler regions condenses.

An energy balance for such an event shows that the kinetic energy of fission fragments and neutrons is about 7.45×10^{13} J; that released by prompt photons and the absorption of excess neutrons is about 0.62×10^{13} J; that from the long-term decay of fission products about 0.44×10^{13} J; and about 0.49×10^{13} J is released harmlessly as neutrinos. Thus, about 8.5×10^{13} J is available as dissipatible energy. About half of this energy is dissipated in the shock wave, which initially travels above the speed of sound, and slows after about 2 km to the speed of sound. Air is accelerated by the passage of the shock wave to velocities of the order of 500 m/s (1 800 km/h) close to the event and falls to about 20 m/s at a distance of 3 km. The blast wave so created has a duration of about one second and gives rise to large forces. Pressures as high as 350 kPa are achieved 500 m from the event, and fall to about 100 kPa at a distance of 1 km, to 50 kPa at 1.6 km and to 10 kPa at 3.6 km.

Shock effects are magnified by the Mach effect. If the bomb explodes in the air, the primary shock wave is reflected from the ground and the reflected wave interferes constructively with the primary wave. Thermal energy in the infrared region comprises about 35% of the total energy released, which, for a fireball of the order of 100 m in diameter, at an average temperature of about 5 000 K and with an emissivity of about 0.6, would imply a peak emission at about 1 nm and a total dissipation in the ultraviolet to far infrared portion of the

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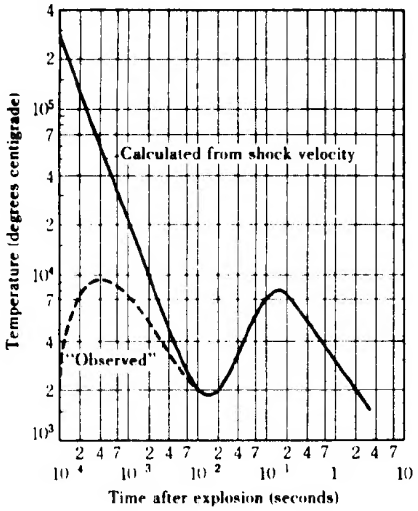

Fig. 2. Change in apparent fireball surface temperature with time, for a 20 kt weapon.

spectrum of about 45% of the total energy. Some energy is released as prompt gamma rays and neutrons. It appears that this is about 3% of the total energy, but varies significantly with the construction of the weapon. The remaining energy is dissipated largely by delayed beta and gamma X rays from the fission products.

To summarize, therefore, a nuclear bomb of 20 000 tons of TNT equivalent dissipates its energy approximately as follows:

Blast propagated by the shock wave	4.2×10^{13} J
Thermal, visible and near UV electromagnetic energy	3.6×10^{13} J
Prompt gammas and neutrons	0.3×10^{13} J
Fission product decay	0.4×10^{13} J
Neutrinos	0.5×10^{13} J
Total	9.0×10^{13} J

Short-term effects of energy released upon the environment

Close to the hypocentre, blast effects are largely vertical; further away, the total effect is reduced but the effects are increasingly in the horizontal direction. The results may be summarized as follows:

Distance from hypocentre (km)	*Damage*
0.5	Most buildings almost totally destroyed.
0.8	Reinforced-concrete, earthquake-proof buildings begin to remain structurally repairable.
1.6	Multistorey brick buildings destroyed almost completely.
2.6	All buildings require repair before use; wooden buildings structurally unsound.
3.2	Wooden buildings begin to be structurally repairable.
3.6	All windows broken; moderate plaster damage.

The vertical nature of the forces close to the hypocentre is evident from Fig. 3, which shows the ruins of the Hiroshima Gas Company's building, which had a reinforced-concrete frame and brick curtain-walling some 250 m from the hypocentre.

The thermal energy is attenuated with distance for two reasons, the familiar inverse-square relationship, and the absorption and scattering of electromagnetic radiation in the atmosphere. On the day the bo[m] was dropped, the sky was clear ov[er] Hiroshima, and visibility was greater th[an] 20 km, so the second effect was limited. Assuming that the visibility was exactly 20 km, the following are the thermal energy fluxes which would have been experienced.

Distance from hypocentre (km)	*Thermal flux (MJ/m²)*
0	4.2
0.5	2.2
1.0	0.9
1.5	0.4
2.0	0.2
3.0	0.1

Such thermal fluxes were sufficient to raise temperatures at the hypocentre to between 3 000° and 4 000°C. One kilometre from the hypocentre, roof tiles were vaporized (Fig. 4), indicating temperatures of over 1 800°C. Three kilometres from the hypocentre, trees and wooden poles were charred on the surface facing in the direction of the burst. Widespread conflagration was another result, and a firestorm grew. From 11h00 to 15h00 there were local winds of 18 m/s as cold air was drawn in a whirlwind to the focus of the storm. By 17h00 all fuel within a 2 km radius of the hypocentre had been consumed, and the wind died. The ashes from this fire were drawn up by buoyancy forces, mixed with the residue of the explosion, and were precipitated as 'black rain' as the moisture condensed.

The high energy photons (gamma rays) from the bomb had little immediate effect upon the environment except to excite secondary fluorescent X rays. The neutrons, which were largely fast (with a velocity of over 10^6 m/s), interacted with other matter and gave rise to induced radioactivity. The approximate dose rates were:

Distance from hypocentre (km)	*Gamma-ray dose (rad)*	*Neutron dose (rad)*
0	1×10^4	2×10^4
0.5	2×10^3	4×10^3
1.0	3×10^2	2×10^2
1.5	2×10^1	1×10^1

Certain nuclides in particular were activated, namely $^{56}_{25}$Mn, $^{24}_{11}$Na, $^{46}_{21}$Sc and $^{60}_{27}$Co, as shown in Fig. 5. As late as 1962, $^{60}_{27}$Co could still be detected in samples of iron collected in the vicinity. Also important in the short-term dose calculations was $^{28}_{13}$Al, with a half-life of only about 2.3 minutes. Immediately after the explosion, the activity

Fig. 3. The three-storey Hiroshima Gas Company's building 250 m from the hypocentre.

one metre above the soil due to $^{28}_{13}$Al was about 200 times that due to $^{56}_{25}$Mn.

This radiation had, in general, no detectable effect upon fauna observed in the immediate vicinity. Bombardier beetles, earwigs, beach fleas, earthworms, tiger beetles, moths, mosquito larvae, rats, rabbits and mice showed no effects. Some *Drosophila* mutations were observed, but it could not be concluded these were the direct effect of the radiation. Many fish died, but this seemed to be the effect of blast rather than of radiation. Birds suffered from blast and burns. A number of horses clearly showed both thermal and radiation injuries. The major effects upon plants were those of blast and heat. However, the stumps of trees within 700 m of the hypocentre tended not to bud at first. There was retardation of growth, fasciation, malformation and variegation of herbaceous species. These effects disappeared within three years after the blast. Some short-lived chromosomal abnormalities were observed.

Fig. 4. A roof tile with clear evidence that its surface temperature exceeded 1 800°C during the 0.3-s thermal pulse caused by the explosion.

Long-term effects upon the environment

The longer-term effects of the two nuclear weapons upon the environment were small, though detectable. The physical damage was made good with remarkable speed. By 1948, houses and buildings were rapidly increasing in number so that it became difficult to study long-term effects upon vegetation. The primary long-term effect was that of the longer-lived radionuclides. Some of these consequences were noted above (Fig. 5). Ashes and nuclides from the

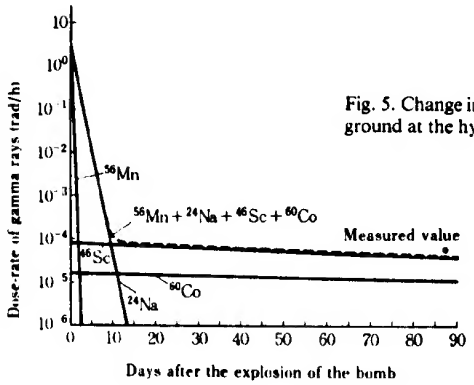

Fig. 5. Change in dose rate with time, one metre above the ground at the hypocentre, Hiroshima.

explosion were carried over an elliptical area, 19 km long and up to 11 km wide, to the north-west of the hypocentre at Hiroshima. At Nagasaki, rainfall 3 km to the east of the hypocentre played a major part in removing activity from the atmosphere. The area around the Nishiyama reservoir was particularly severely contaminated.

Two months after the explosions, the fallout contributed up to 0.05 mrad/h at Hiroshima and up to 1 mrad/h at Nagasaki. The integrated dose over 35 years, from one hour after the explosion to 1980, in the air one metre above the ground, amounted to a maximum of between 4 and 40 rad at Hiroshima and between 50 and 150 rad at Nagasaki. It should be recalled that the maximum permissible dose for members of the general public should not exceed 5 rad/yr, or 175 rad over 35 years, for those of 18 years of age and older. The ALARA ('as low as reasonably achievable') principle should obviously apply, but it is interesting that the integrated dose from fallout after such devastating events should only approach the 'tolerable' level.

Short-term effects upon the populace

It was difficult to assess the short-term effects of the explosions on people. The total destruction of the cities made accuracy impossible. Figure 6 illustrates the acute casualty and mortality levels at Hiroshima and Nagasaki, and Fig. 7 indicates that in both cities the death rate was halved every six days after the explosions. Within forty days most of the acute effects had passed. After a year, the casualties at Hiroshima were as shown in Table 1.

Thermal injury was the primary cause of this disaster. Recall that thermal fluxes of over 0.9 MJ/m² occurred within 1 km of the hypocentre. Such fluxes, acting on bare skin for 300 ms, will result in charring and carbonization. Even fluxes of 0.1 MJ/m², 3 km from the hypocentre, will cause erythema of the skin within 300 ms. Such fluxes are unusual in thermal injury, and the injuries are known as 'flash' burns. Shielding is obviously possible, and in fact reduced the mortality rate at a distance of 2 km by a factor of about five. The usual thermal radiation phenomena were observed, with light-coloured materials reflecting the heat and offering better shielding than dark-coloured materials.

Another important cause of death was blast injury. There were primary injuries due to the blast, but the effects were masked by the thermal injuries. Primary visceral injuries typical of conventional blasts were relatively rare, and the incidence of blast injury was higher in those who had been indoors rather than outdoors. This suggests that secondary injuries from materials accelerated by the blast were the main cause of fatalities. The survivors hospitalized after the blast on 13 August showed the following injuries: thermal burns, 50.2%; trauma, 33.3%; both thermal burns and trauma, 16.5%. The traumas were mainly contusions (54%) and wounds due to glass fragments (35%). The latter were particularly distressing, as they were often the result of a myriad of glass splinters embedded in the body.

Finally, there were the immediate deaths caused by high-energy radiation. It will be recalled that about 0.3×10^{13} J was dissipated as prompt gamma rays and neutrons, which resulted in in-air doses of greater than 300 rad of gamma radiation and 200 rad of neutrons within 1 km of the hypocentre. At the hypocentre, the doses were 10 000 rad of gamma rays and 20 000 rad of neutrons. A whole-body dose of 10 rad has a barely detectable effect; one of 100 rad causes mild acute symptoms, with some diminution of the white cell counts; one of 1 000 rad depresses blood cell and platelet formation, damages the intestine, and usually leads to death within 30 days; a whole-body dose of 10 000 rad leads to immediate signs of damage to the central nervous system, with death within a few hours.

Table 1. Total casualties at Hiroshima, 6 August 1945 to 10 August 1946 (military personnel excluded).

Distance from hypocentre (km)	Killed	Severely injured	Slightly injured	Missing	Not injured	Total
Under 0.5	19 329	478	338	593	924	21 662
0.5 – 1.0	42 271	3 046	1 919	1 366	4 434	53 036
1.0 – 1.5	37 689	7 732	9 522	1 188	9 140	65 271
1.5 – 2.0	13 422	7 627	11 516	227	11 698	44 490
2.0 – 2.5	4 513	7 830	14 149	98	26 096	52 686
2.5 – 3.0	1 139	2 923	6 795	32	19 907	30 796
3.0 – 3.5	117	474	1 934	2	10 250	12 777
3.5 – 4.0	100	295	1 768	3	13 513	15 679
4.0 – 4.5	8	64	373		4 260	4 705
4.5 – 5.0	31	36	156	1	6 593	6 817
Over 5.0	42	19	136	167	11 798	12 162
Total	118 661	30 524	48 606	3 677	118 613	320 081

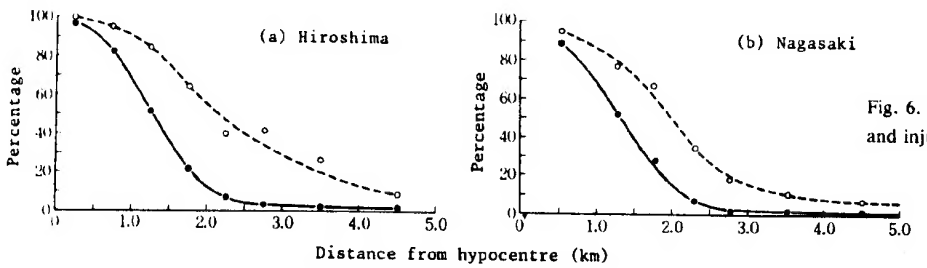

Fig. 6. Variation of acute mortality (●—●) and injury (o—o) with distance.

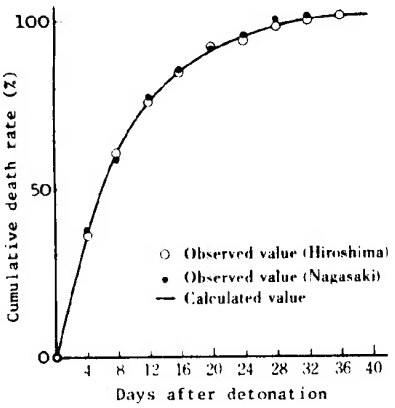

Fig. 7. Change in relative death rate with time.

In the vicinity of the hypocentre, there were clear signs of people having suffered massive radiation doses. Over 30% of the eventual survivors and 16% of those who soon died were highly nauseous within a few hours, and many suffered diarrhoea often accompanied by internal haemorrhage within a few days. Deaths among otherwise only mildly injured victims occurred seven to 10 days after the explosion, usually with signs of cerebral damage. Autopsies were carefully performed on 25 who died during this acute phase; of these, 19 died primarily from thermal injuries. There was, however, evidence of radiation damage in most cases. At this acute stage, it showed itself primarily in the haematopoietic organs, particularly the spleen, where lymphocytes were almost entirely absent. About two weeks after the blast, the acute phase of injury drew to a close. About 90% of the eventually fatal cases had died by this stage, and about 90% of the survivors who received medical treatment during this phase complained of thermal injury.

Intermediate-term effects upon the populace

From the beginning of the third week after the explosions to the end of the eighth week, the effects of radiation took their toll, and a further 10% of the ultimate total of fatal cases died during this period. The first sign of the effects of radiation was usually the loss of hair, or epilation, the degree of which varied with distance from the hypocentre and with shielding. At Hiroshima, 76% of those exposed within 1 km of the blast, who were either outside or in wooden buildings, suffered epilation. The onset was 17 days after the explosion, on average, and continued for one to two weeks. Hair regeneration then started after 8 to 10 weeks.

The second sign was subsurface haemorrhage, which showed itself as purplish spots or patches. The incidence among those exposed within 1 km was about 50%. There were related oropharyngeal lesions, the incidence of which was highly correlated with epilation to about 1.5 km from the hypocentre; at greater distances, the lesions were more common, and were as high as 10% at 4.5 km. Other haemorrhages were common, particularly nasal and uterine. Ocular haemorrhage was also prevalent. All these signs were often aggravated by fever, which rose as each new symptom appeared and continued until death intervened.

Haematological examination showed that damage to the bone marrow had reduced the regeneration of blood cells, particularly white cells. Red cell counts were typically 80% of normal, haemoglobin 50% of normal, and white cells about 5% of normal, in those exposed to 450 to 600 rad and who died within 14 days. In less severe cases, about half the population died within 40 days; their white cell count was never less than about 10% of normal. A third group had a minimum in their white cell count after about four weeks, at about 25% of the normal count; about 10% of this group died, usually more than 40 days after the explosion and of complications rather than from direct injury due to radiation.

There were those who were potentially affected by secondary radiation, i.e. by the radiation from neutron-induced radioisotopes in the air and soil, and by fallout. Those entering Hiroshima an hour after the explosion and staying near the hypocentre for five hours would have received about 20 rad; the next day they would have received less than 10 rad in eight hours. In such populations, about one-third had less than 80% of the normal white cell count. Those exposed to fallout experienced marked malaise, headache and abnormal menstruation soon after the event. In the longer term there was a marked *increase* in the white blood cells, particularly leukocytes, with counts over five times the normal in extreme cases, 50 to 80 days after the explosion. This effect occurred and disappeared most rapidly in children. Eight years after the explosion the white cell count was almost normal in the population living in the areas most affected by fallout.

Long-term effects

A primary effect was the formation of scar tissue at the site of thermal injury, which led to deformity or functional disturbance. Particularly distressing was the formation of 'keloids', or the overgrowth of scar tissue during regeneration, forming irregular protrusions like the shell of a crab. This occurred in about two-thirds of all thermal burns, usually within 150 days of injury and gradually disappeared over the next decade or so.

The blood disorders so characteristic of radiation injury persisted for several years. A year after the explosion, there was a high incidence of general malaise and dizziness associated with some reduction in the red and white cell counts. After two and three years these symptoms persisted, but to an ever smaller degree. After eight years anaemia was a statistically significant experience, and this persisted, while declining, for up to 15 years.

The formation of cataracts in the eye was common. Those exposed while infants and examined 14 years later had a greater than 50% incidence if exposed within 1 km of the hypocentre, and a greater than 10% incidence if exposed within 1.4 to 1.6 km, although in only 13% of all cases was the size of the cataract moderate (no marked or severe cases were found).

Amenorrhea was common (greater than 50% incidence) after the explosion and typically continued for five to six months. The majority of women over 45 went into menopause. Exposure to radiation did not appear to affect the age of menarche in teenage girls. There was no evidence for an increase in sterility due to exposure, but there was a statistically lower conception potential in exposed women from Hiroshima than in those from Nagasaki. The evidence for an increase in the number of stillbirths with an increase in exposure to radiation, or changes in sex ratio or birthweight, was inconclusive. There was some evidence for an increase in the incidence of malformations among those born of exposed women, particularly in the bone, heart and large vessels, but no malformation was characteristic, and the increase in incidence lessened with time.

Exposure *in utero* led to increased foetal mortality from about 3% in the unexposed to over 20% in those exposed within 2 km of the explosions. Neonatal and infant death rates were about 4% in the unexposed and over 25% in those significantly exposed within 2 km. In a 24-year study of nearly 1 300 children from both Nagasaki and Hiroshima who had been exposed to some radiation *in utero*, there was a significant increase in mortality, particularly up to the age of seven. At the age of 17, there were significant differences in mass, height, and head and chest circumferences for those exposed *in utero* within 1.5 km.

A particular problem has been the development of microcephaly (a head circumference more than two standard deviations below the age- and sex-specific mean size). The normal incidence was about 3%; among those exposed within 1.5 km, the incidence was about 25% (30 cases, of which 12 were severe). Severe mental retardation often accompanied this. Microcephaly was severest in those exposed during the first trimester.

I turn now to what might be termed the very long-term effects, as studied in a population of about 109 000 since 1950. The population is distributed into various radiation dose categories as shown in Table 2. The control group matches the exposed

Table 2. Radiation dose categories of the population at risk.

			Tentative dose in 1965 (rad)						
	Total	Nonexposed	0	1-9	10-49	50-99	100-199	200+	Unknown
Hiroshima	82 085	20 176	29 943	13 787	10 707	2 665	1 677	1 460	1 670
Nagasaki	26 682	6 347	4 699	6 705	3 700	1 231	1 229	1 310	1 461
Total	108 767	26 523	34 642	20 492	14 407	3 896	2 906	2 770	3 131

group as nearly as possible in the distribution of age and sex. About 25% of this population died between October 1950 and September 1974. A comparison of the causes of mortality between the unexposed and the 200+ rad group is made in Fig. 8. There was a very significant increase in the incidence of leukemia and a significant increase in non-leukemia cancers and disease in the exposed sample. However, it is noteworthy that the greater mortality due to the increase in cancers was not unduly severe. Figure 9 gives the excess deaths from cancers per annum per million of population, for those exposed to more than 100 rad. There were about 145 cases of leukemia amongst survivors who received 100+ rad, over the period 1946 to 1975. It may also be noted that early entrants to the radioactive cities showed no long-term effect due to the induced radioactivity.

Other cancers which may well exceed the background level include cancer of the thyroid, lung, breast (in women) and salivary glands. Again, however, the numbers involved must be noted. For instance, in a study of over 10 000 women during the period 1958 to 1966, there were 22 definite cases of breast cancer observed where 22 had been expected. However, in the control group of nearly 2 500, only two were observed whereas 5.4 had been expected; and in the group of nearly 1 000 who had been exposed to 200+ rad, five were observed whereas two had been expected in the absence of any radiation effect. The possibility that there is also an 'observer' effect due to the intensity of study cannot be ruled out. However, in another study of over 1 000 women exposed to 100+ rad between the ages of 10 and 19, there were 10 cases of breast cancer up to 1969, whereas the expected number in another control group of the same age distribution and over the same period was only 1.4. It thus seems likely that there is a correlation between the degree of exposure and the incidence of breast cancer.

While the evidence linking a particular cancer and exposure to massive doses of radiation may be equivocal, there is no doubt that chromosomal changes occur. Moreover, the number of aberrations is directly related to radiation dose, although at Hiroshima the relationship was linear and at Nagasaki it was quadratic. Furthermore, symmetric aberrations and dicentric/acentric aberrations have persisted from the time of exposure to the present. However, there is no evidence for a link between chromosome aberration and disease: 'Chromosome aberration exists as if it were unrelated to health but is closely related only to radiation dose' (p. 319).

There remains the fear that such chromosomal aberrations may be transmissible to children born of irradiated parents. The possibility of this occurring has been the subject of the most searching enquiry, but 'genetic surveys undertaken to date have yielded no positive evidence for a genetic hazard due to atomic bomb radiation' (p. 326). There may, however, be reasons for supposing that any effect would show itself less readily in the human population than it would amongst laboratory populations of mammals (where it is readily seen).

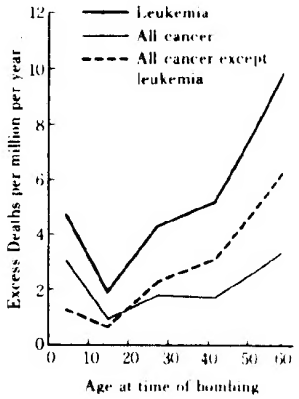

Fig. 9. Absolute risk of cancer-caused death according to age at time of exposure to 100+ rad.

Discussion

At this point in the book, the tenor of the text changes. The full description of the quantitative findings, summarized above, comes to an end, and consideration is given to the sociological consequences of the bombing.

Similarly, this review must now change from a bald presentation of the facts to a personal interpretation of their significance. Of course, even the summary has been somewhat subjective — some pieces of evidence have been more strongly accented than others. Equally, this happened in the original — it would not be possible to condense some 800 primary references without a degree of subjective selection. However, the reader should be warned that in what follows, the interpretation is personal and based upon the selection of evidence presented in the previous sections.

I suggest that the common wisdom assesses the hazard of nuclear weapons as being that of 'radiation' — specifically, high-energy electromagnetic radiation. There is a widespread belief in reproductive sterility being one consequence of their use, and where that does not occur, in the production of malformed offspring. There is also a common belief in a consequential massive increase in cancers and a host of other ill-defined illnesses. Many people con-

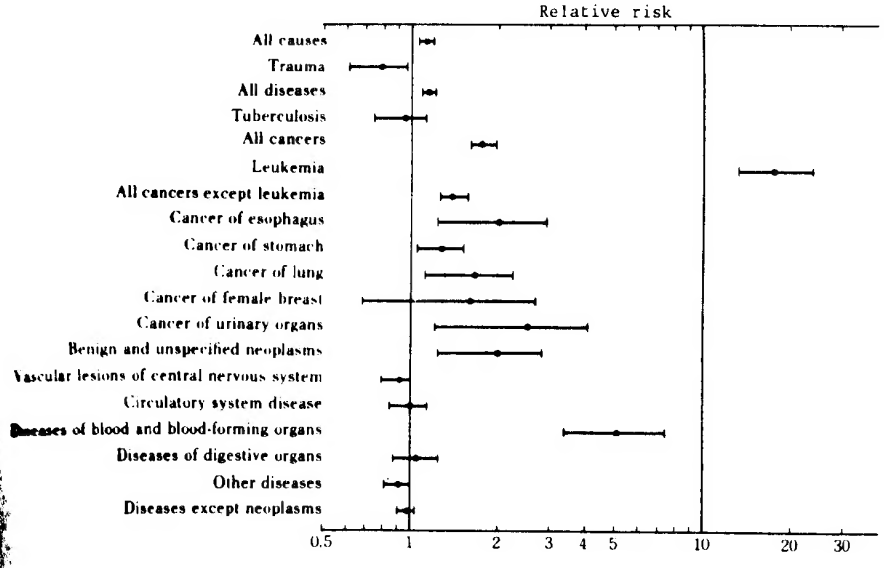

Fig. 8. Causes of death of survivors of explosions who were exposed to 200+ rad, compared to those exposed to normal environmental radiation, in the period 1950 to 1974. Bars represent 80% confidence interval.

sider that the whole face of the world will be changed by invisible radiation, and much of the attack upon the use of nuclear weapons is based upon this. (The production of electrical energy by controlled nuclear fission is perceived by some as subject to a similar hazard, and is vigorously opposed for this reason.)

This is not the place to summarize the evidence for these points of view. It suffices to say that they exist and are a major element in opposition to the use of nuclear weapons on the one hand, and obstacle to the growth of the nuclear energy industry on the other. I submit that the attack upon the use of nuclear weapons would be far more successful, and far more soundly based, if the parallels between these weapons and those which society already finds intolerable were drawn. Specifically, there is a consensus that chemical warfare is intolerable. Chemicals are known which could cause devastation on a scale similar to that which occurred at Hiroshima and Nagasaki. Seven years after a slight industrial accident at Seveso, in northern Italy, the surrounding countryside is uninhabitable, and attempts to dump contaminated soil became a major political issue in the European Economic Community earlier this year. But what could cause greater distress than the chemical changes which occur when the human body is exposed to thermal fluxes of over 0.1 MJ/m^2? At both Hiroshima and Nagasaki, these chemical changes were responsible for the deaths of about one hundred thousand people within a few days. And these were civilians in a well-nigh defeated country, not combatants locked in a Somme salient.

Fortunately, mankind has perceived the problem of heat damage to civilians. The events of the Vietnam war, and the disastrous effects of the indiscriminate use of napalm there, are fairly clear. The definition of chemical weaponry is slowly being extended to cover this awful weapon, too. However, the thermal flux from nuclear weapons has been overlooked, even though it is far more terrible in its effect than a few hundred megajoules of naplam.

Similarly, mankind has generally reacted against the use of bullets which expand on contact. Even the preparation of a 'dum-dum' bullet can be treated as a criminal act. Yet there appears to be no realization that nuclear weapons can accelerate many strange shapes to bullet-like velocities, with results equivalent to those of the worst shape of bullet.

Then there are the short-term effects of massive doses of radiation, which, at much over 1 000 rad, act upon the central nervous system as terminally as the most potent of the banned nerve gases, and which, at much over a few hundred rad, lead to effects upon the body as pernicious as the worst of the forbidden biological weapons. It is a most challenging question, why one weapon can be rejected and yet another, with an equivalent or greater effect, can continue to be considered for possible use.

One thing is certain, however. It is that fallout and induced radioactivity are poor grounds for attacking nuclear weapons. In the first few days after the atomic bombs were used against them, 60 000 Japanese died, primarily of thermal burns; a further 60 000 died over the next few weeks, largely from burns and partially from massive radiation doses. Thereafter, all the delayed effects, all the old wounds aggravating old age, all the cancers, all the tragedies of the exposed unborn, may have accounted for only a further 2 000 deaths. There are no longer any signs of these events in the bodies of those in the path of the fallout, nor any evidence that those who entered the inferno shortly after the explosion were significantly injured. However, though we have moved more than half a lifetime from the most ignoble of experiments, the harrowing memory of its devastation has permanently scarred our minds. Irrational or not, the terror of the invisible, of radiation, remains. To light a star on earth is to incinerate, to immolate, and to irradiate. Of these, the last is to be the least feared.

1. *Hiroshima and Nagasaki; The physical, medical and social effects of the atomic bombings*. Committee for compilation of materials on damage caused by the atomic bombs in Hiroshima and Nagasaki. Hutchinson, London; 1981.

Towards the Production and Maintenance of Disease-free Laboratory Animals

Animal studies are complicated by variability in the response of animals to experimental treatments. This variability interferes greatly with the interpretation and predictability of results. It can arise from a wide variety of factors such as genetic background, nutrition, animal care, environmental effects and disease. An increasing awareness of the importance of these sources of variability in animal studies over the last decade, has led to the establishment of several modern laboratory animal facilities in South Africa. One of their main functions is to produce genetically defined, specified pathogen free (SPF) animals and maintain them under standardised conditions. A major challenge in this endeavour is the production by hysterectomy of SPF rodents and rabbits under barrier conditions.

Conventional laboratory animals maintained in non-barrier buildings harbour a wide range of pathogens, including ecto and endo-parasites, fungi, protozoa, bacteria and viruses, which can either singly or in concert interfere with experimental results by causing clinical or sub-clinical disease in animal colonies. Laboratory animals can be freed of these pathogens by the removal of full-term foetuses from an infected animal by hysterectomy and transfer to the protected environment of flexible, clear plastic film isolators for rearing either by hand feeding or foster mothers.

Interest in isolator technology for the establishment of pathogen-free animals has grown rapidly in South Africa over the last two years and isolator units have been established by the universities of Stellenbosch, Pretoria, Cape Town, and the Witwatersrand, and the State Vaccine Institute at Pinelands in the Cape.

With the commissioning of the new Medical School Animal Unit at the University of the Witwatersrand and installation of isolator equipment, a workshop on isolator technology with practical exercises was held under the auspices of the South African Association for Laboratory Animal Science on 23 – 24 February this year. The workshop was run by Mr J. von Stenglin of the Metal-Plastic company. It was attended by delegates from university animal units, the South African Institute for Medical Research, South African Medical Research Council, the National Institute for Virology, the Animal and Dairy Science Research Institute and the Technikon RSA.

The course reviewed the historical developments of isolator technology from its beginnings in 1895, when von Nuttal and Theirfelder were successful in challenging Louis Pasteur's belief that animal life could not be sustained without bacteria by producing and maintaining a germ-free rat in an unwieldy and complicated isolator system. It examined the wide range of isolator applications for protecting animals and patients from harmful infective gents. Practical exercises were carried out with positive and negative-pressure isolators, which involved the preparation of isolator components, setting up and leakage testing, sterilization of equipment and supplies, transfer procedures for supplying food and water and removing animal wastes, and microbiological screening. Techniques for obtaining germ-free animals by hysterectomy and caesarian section in surgical isolators linked to animal isolators were also demonstrated and discussed.

The course, the first of its kind in this country, has provided a sound foundation for the establishment and development of animals reared in isolators in South Africa, which is the key to providing disease-free animals for teaching and research. **J. Austin**